

Set Name Query
side by side**Hit Count Set Name**
result set*DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ*

<u>L3</u>	(cd18 or 'anti-cd18' or h52) same (infarct or ischemi\$ or stroke)	42	<u>L3</u>
<u>L2</u>	(cd18 or 'anti-cd18' or h52) same (infarct or ischemi\$)	31	<u>L2</u>
<u>L1</u>	bednar-martin\$	9	<u>L1</u>

END OF SEARCH HISTORY

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Term	Documents
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(L4.CLM.).USPT,PGPB,JPAB,EPAB,DWPI.	3

Database:

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<u>L6</u>	L4.clm.	3	<u>L6</u>
<u>L5</u>	(h52) same (infarct or stroke or ischem\$)	1	<u>L5</u>
<u>L4</u>	(cd18 or 'anti-cd18' or h52) same (infarct or stroke)	25	<u>L4</u>
<u>L3</u>	(cd18 or 'anti-cd18' or h52) same (infarct or ischemi\$ or stroke)	42	<u>L3</u>
<u>L2</u>	(cd18 or 'anti-cd18' or h52) same (infarct or ischemi\$)	31	<u>L2</u>
<u>L1</u>	bednar-martin\$	9	<u>L1</u>

END OF SEARCH HISTORY

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L4: Entry 18 of 25

File: USPT

Jan 13, 1998

DOCUMENT-IDENTIFIER: US 5708141 A

TITLE: Neutrophil inhibitors

Other Reference Publication (154):

Winkist et al., "An Anti-CD18 MAb Limits Infarct Size in Primates Following Myocardial Ischemia and Reperfusion", Abstracts of the 63rd Scientific Sessions, III-70 at abstract 2785 (1990).

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L4: Entry 19 of 25

File: USPT

Feb 15, 1994

DOCUMENT-IDENTIFIER: US 5286718 A

TITLE: Method and composition for ameliorating tissue damage due to ischemia and reperfusion

Brief Summary Text (16):

A variety of agents have been identified through preclinical investigations which appear to have the potential to provide some benefit in respect to reperfusion injury. Unfortunately, when applied to the human clinical setting, the results have been disappointing. SOD performed poorly at reducing myocardial infarct size in the clinical setting. A short half-life, poor tissue distribution, and consequently primarily a protective effect on vascular endothelium restricted clinical utility. Efforts to improve the half-life of SOD by forming the polyethylene glycol conjugate did not improve protective activity. Allopurinol has demonstrated some efficacy in early human studies involving renal transplants. Other agents entering human clinical trials include a monoclonal antibody to the neutrophil adhesion molecule CD18, a complement receptor antagonist, fluorinated hydrocarbons, and adenosine or adenosine agonists. However, these and other therapies under consideration have yet to exhibit the desired clinically useful attributes.

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